## TEMPORAL LOBE EPILEPSY: PERSONAL OBSERVATIONS\*

## ROBERT J. STROBOS

Associate Professor and Chief, Section of Neurology New York Medical College, New York

The term "temporal lobe epilepsy" is well established and has much merit. There is a significant correlation between psychomotor seizures and abnormal temporal lobe discharges in the electroencephalogram. The typical aura of a patient's seizures may be reproduced by electrical stimulation of the temporal lobe. Seizures have been abolished by temporal lobectomy, and the removed temporal lobe may contain a clearly discernible focal lesion which easily could have caused the convulsions.

Consequently, it is often implied that temporal lobe epilepsy, or indeed psychomotor epilepsy, is caused by focal lesions in the temporal lobe. This conclusion is not justified.

First, it is important to differentiate between temporal lobe epilepsy and psychomotor seizures. Not all psychomotor seizures are caused by a temporal lobe lesion: some psychomotor seizures are accompanied by bilaterally synchronous discharges in the electroencephalogram without any focal features in the record. In a series of patients with centrencephalic epilepsy, 17 per cent had seizures consisting of transient and paroxysmal episodes of confusion, which clinically would have to be classified as psychomotor attacks. It may well be that the epileptic discharge in such cases predominantly involves the temporal lobes and connected subcortical structures, but there certainly is no evidence that the seizures originate in, or are caused by a focal temporal lobe lesion.

Furthermore, it is important to keep in mind that temporal lobectomy, in epileptic patients with well localized abnormal discharges in the temporal region, results in cessation of seizures in only about 50 per cent of the cases. After temporal lobectomy, new discharging foci may appear along the margins of the ablation, or spiking in other locations may occur at a later date.<sup>2</sup> Thus, the possibility exists that the abnormal discharges recorded over the temporal lobe in some cases do not indicate

<sup>\*</sup> Presented as part of a Symposium on Temporal Lobe Epilepsy, held at a combined meeting of the New York Neurological Society and the Section on Neurology and Psychiatry, The New York Academy of Medicine, November 9, 1961.

a focal lesion in the temporal lobe itself, but are projected from a distant source. "Mirror-foci" are now well recognized in electroencephalography; abnormal impulses from one area may trigger off abnormal discharges in a distant, but connected area. There was good electroencephalographic evidence in a few of my cases that the temporal lobe focus was triggered from a distant focus, and that isolated, focal temporal lobe spiking occurred transiently in patients with centrencephalic epilepsy.<sup>2</sup> This should not be surprising, as it is well documented that the temporal lobes, including the hippocampus and the amygdaloid nuclei, have a very low threshold for obtaining after-discharges by electrical stimulation.<sup>3</sup>

It should also be emphasized that the temporal lobes fail to reveal a well-demarcated focal lesion in the majority of patients with temporal lobe epilepsy. The significance and specificity of the often reported "atrophic" lesions is still quite uncertain.

These considerations have only theoretical value, as long as we are treating patients with anticonvulsant medication, but they become highly important when selecting patients, or when establishing criteria for surgical intervention.

Temporal lobe epilepsy is primarily treated with anticonvulsant medication. The majority of patients will never be considered for temporal lobectomy. In our own series of long-followed and intensively treated patients only 10 per cent ultimately underwent temporal lobectomy.1 Treatment with anticonvulsant medication completely controlled seizures in 26 per cent, while an additional 27.5 per cent were more than 90 per cent controlled. There were 17.5 per cent who showed more than 50 per cent improvement, the remaining 29 per cent had no appreciable benefit from medication. Thus, 46.5 per cent conceivably could have been candidates for surgery, but many of these were not considered for various reasons: 1) because the disease was not a social handicap (very infrequent seizures; seizures only during sleep; seizures were mild and not easily observed by onlookers); 2) because the findings on electroencephalography made the patient ineligible for surgery (bitemporal independent foci; too many bilateral discharges, even though there was focal preponderance and phase-reversal in one temporal lobe; too much spread to the mid- and posterior temporal region); 3) because of psychologic disturbances in the patients.

Approximately half of the patients who undergo temporal lobec-

tomy are cured of their seizures, and this, of course, is a tremendously gratifying result. There is, however, further need for research in this area. Is there any way in which these results can be predicted, so that indications for surgery may be more sharply drawn? Electroencephalographers have been studying pre-surgery records in cases with favorable and unfavorable outcome in order to sharpen criteria. Clinical research might also be useful. Would it, for instance, be possible to differentiate patients with actual focal temporal lobe lesions from those with referred foci, by the nature of the aura? There is some evidence that an olfactory aura occurs with a higher frequency in the first group.<sup>2, 4</sup>

A great concern in the treatment of temporal lobe epilepsy is the possibility of a brain tumor as the etiologic agent. In our series of temporal lobe tumors, epilepsy was the first appearing symptom in 32 per cent of the cases.4 In a series of cases treated as convulsive disorder for 2 to 16 years before a diagnosis of tumor was made, one-third of the tumors were localized in the temporal region.<sup>5</sup> We also know that surgical specimens of temporal lobectomy not too infrequently contain an unsuspected tumor. Brain tumors are easily diagnosed when there is evidence of increasing neurologic deficit or of increased intracranial pressure, but these manifestations may occur late in the course of the disease in cases of slow-growing tumors. In a study of our material,<sup>5</sup> we found some other criteria that may be helpful in the earlier recognition of such tumors: 1) the seizures are usually not controlled by anticonvulsant medication; 2) a definite change in seizure pattern often occurs in the course of the illness; 3) an original, sharply delineated spike focus is increasingly replaced by focal slow wave activity. It was also noted that an olfactory aura more frequently precedes seizures caused by temporal lobe tumor than seizures associated with a temporal lobe spike focus, where there was no proof of a focal lesion. It may, therefore, be advisable to employ special ancillary studies, such as pneumoencephalography or angiography, when some of these features occur in a patient with temporal lobe epilepsy.

## REFERENCES

- Strobos, R. R. J. Prognosis in convulsive disorders, Arch. Neurol. 1:216-25, 1959.
- Strobos, R. R. J. Mechanisms in temporal lobe seizures, Arch. Neurol. 5: 36-45, 1961.
- 3. Strobos, R. R. J. and Spudis, E. V. Effect of anticonvulsant drugs on cortical and subcortical seizure discharges
- in cats, Arch. Neurol. 2:399-406, 1960.
- 4. Strobos, R. R. J. Tumors of the temporal lobe, Neurology (Minneap.) 3: 752-60, 1953.
- Strobos, R. R. J., Alexander, E., Jr. and Masland, R. L. Brain tumor presenting as convulsive disorder, Dis. Nerv. Syst. 19:518-22, 1958.